

Kidney infections

Acute pyelonephritis

Inflammation of renal parenchyma and renal pelvis

Typically fever, chills and costovertebral angle tenderness +/- LUTS

Gram negative bacteria

E. coli*

Proteus

Klebsiella

Pseudomonas

Enterobacter

Citrobacter

Serratia

Enterococcus, S aureus and S epidermidis occasional GP orgs.

*E. coli expressing Type II p-fimbriae responsible for 80% of cases

Diagnosis

Urinalysis typically positive for nitrites and leucocytes

MSU > 10^4 cfu/ml with pyuria suggestive (?arbitrary definition) – 20% have counts less than 10^5

WBC casts

Elevated CRP and ESR

Imaging

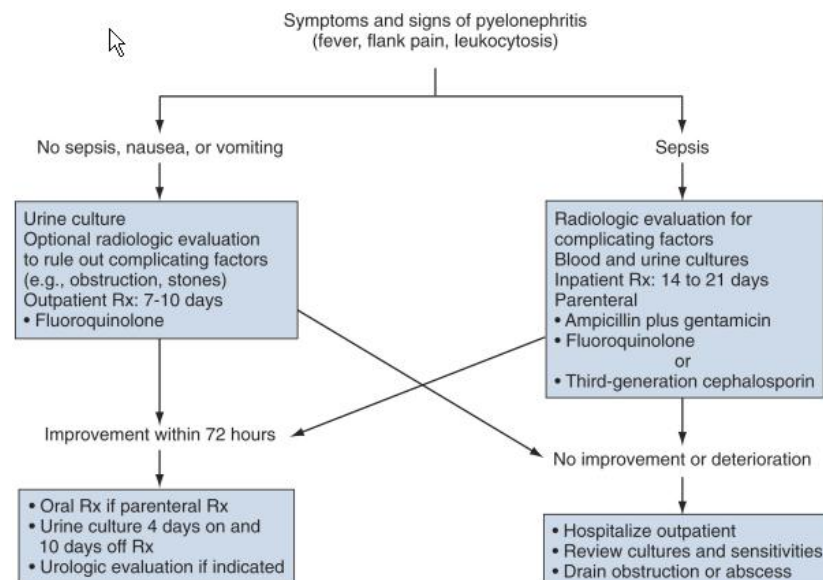
Not indicated in acute uncomplicated pyelonephritis

Reserved for complicated cases or those not responding after 72 hrs

IVU Renal enlargement in 20% if done acutely – may be focal (lobar nephronia) appearing as a mass lesion
Reduced nephrogram and delay secondary to cortical vasoconstriction

USS Excludes obstruction, identifies stones and gross focal changes

CT Excellent for focal changes and identification of gas and stones. Generally reserved for those failing to improve.



Management

Mild, outpatient PO TMP-SMX 14 days
 *PO Ciprofloxacin 500mg bd 7 days
 7 day course of cipro more effective and fewer side effects than 14 day course of TMP-SMX (Talan 2000).
 Third generation cephalosporin cefpodoxime proxetil 200mg bid equivalent efficacy cf. ciprofloxacin 500mg bid.
 No adequately powered studies of penicillin/BLI vs. fluoroquinolone or TMP-SMX

Table 2.4: Oral treatment options of acute uncomplicated pyelonephritis in adult pre-menopausal non-pregnant women according to level of evidence and grade of recommendation. (For parenteral therapy, see text.)

Substance	Dosage	Duration	LE	GR	Author, year	Ref	Remarks
Ciprofloxacin	500 mg bid	7 days	Ib	A	Talan 2000	69	a) Ciprofloxacin significantly more effective than ceftriaxone/TMP-SMX and with trend towards less AE
CiproXR	1000 mg od	7-10 days	Ib	A	Talan 2004	70	b) Efficacy and tolerance of extended release ciprofloxacin (ciproXR) 1000 mg od equivalent with 10-day conventional ciprofloxacin
Cefpodoxime*	200 mg bid	10 days	Ib	B	Naber 2001	73	c) Clinically equivalent with ciprofloxacin 500 mg bid
Gatifloxacin	400 mg od	10 days	Ib	A	Naber 2004	71	d) Equivalent with ciprofloxacin 500 mg bid, not available in Europe
Levofloxacin	250 mg od	10 days	Ib	A	Richard 1998	72	e) Equivalent with ciprofloxacin 500 mg bid
Lomefloxacin	400 mg od	10 days	Ib	B	Richard 1998	72	f) Study statistically underpowered
TMP-SMX	160/800 mg bid	14 days	Ib	B	Stamm 1987 Talan 2004	68 70	g) Only if uropathogen is known to be susceptible to TMP

*Cefpodoxime proxetil.

LE = level of evidence; GR = grade of recommendation; TMP = trimethoprim; SMX = sulphamethoxazole; tid = three times daily; bid = twice daily; od = once daily; AE = adverse events.

Mod/severe, I/P IVI, IVABx, antiemetics, painkillers 14-21days
 IV Gent and ampicillin (7mg/kg; 1g qds)
 IV Ceftriaxone 1-2g qds
 IV Ciprofloxacin 400mg bd

Pregnant IV Ceftriaxone 1-2g qds
 IV Gent and ampicillin
 IV Tazocin 4.5mg tds
 IV Imipenem 500mg qds

Imaging for complicating factors

10-30% relapse rate after 14 days appropriate Rx – repeat MSU 4 days on and 10 days off Rx. Usually cured after further 2 week course.

Acute focal bacterial nephritis

Uncommon form of acute pyelonephritis

Similar presentation but more unwell

Often in diabetics and immunocompromised

Mass on USS

Wedge shaped area of poor perfusion on contrast enhanced scan

Management as for acute pyelonephritis

Emphysematous pyelonephritis

Rare severe necrotising infection of kidney; a/w mortality rates of 20-43%

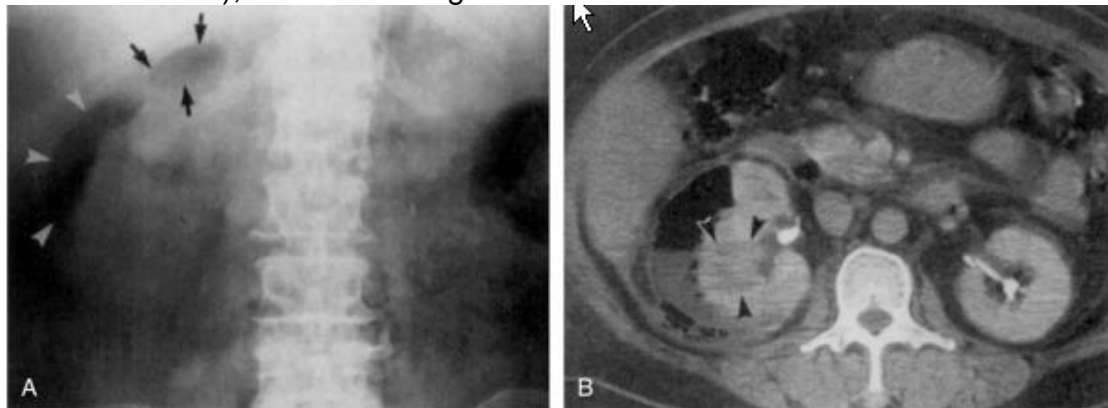
Usually in diabetics (70-90%), always in adults, often with a history of UUT obstruction

Classic triad of fever, loin pain and vomiting always seen; 50% palpable mass, occasionally with crepitation

E Coli is usually responsible – produces carbon dioxide from metabolism of sugars

Diagnosis

AXR shows gas in renal parenchyma in 85% cases (mottled vs. crescent), but CT investigation of choice



Classification

Divided into type 1 and type 2 based on CT findings:

- | | |
|--------|---|
| Type 1 | Generalised renal infection
Gross parenchymal destruction
Small gas locules throughout kidney
Minimal fluid collection
~60% mortality |
| Type 2 | Localised renal infection
Mild/moderate focal destruction
Coalescence of gas focally
Air-fluid level
~20% mortality |

Management

- Resuscitation
- Broad spectrum antibiotics
- IV insulin sliding scale
- Emergent nephrectomy (medical therapy alone a/w 60-80% mortality)

Renal abscess

Pus in the renal parenchyma

Organisms

- (i) Gram-negative
 - Most common
 - Almost universally from ascending route
 - Two-thirds in association with stones or renal damage; occasionally a/w reflux disease
 - Experimental and clinical data suggest haematogenous seeding by GNB rare, except in instances of complete collecting system obstruction
- (ii) Gram-positive
 - Uncommon

Historically majority of renal abscesses were secondary to haematogenous seeding by staphylococci. Now rare, except for immunocompromised and IVDU

(iii) Mycobacteria

Very rare even in patients with genitourinary TB

Usually parenchymal scarring, wall thickening and calcification, hydronephrosis and hydrocalycosis

Isolated hydrocalycosis may be mistaken for renal abscess however

Clinical features:

Fever, chills, rigor

Malaise and lethargy

Abdominal/flank pain

Leucocytosis

Positive UTI (GNB only)

Diagnosis:

USS

Initially indistinct echopoor parenchymal SOL with varying degree of internal echoes

maturation into discrete lesion of variable echotexture with hyperechoic margin

Presence of air casts dense echo/shadow

CT

Renal enlargement

Discrete low attenuation mass with surrounding rim of higher attenuation (ring-sign)

Management:

Dependent on size

Small abscesses $\leq 3\text{cm}$ a/w resolution with prompt administration of appropriate ABx

Abscesses $> 3\text{cm}$ suitable for percutaneous drainage.

Failure to respond to above should prompt a search for perinephric abscess

Surgery may be considered as primary surgery for abscesses $> 5\text{cm}$

Infected hydronephrosis/pyonephrosis

Definitions below from Campbells

Infected hydronephrosis – bacterial infection in hydronephrotic kidney

Pyonephrosis – infected hydronephrosis with suppurative destruction of renal parenchyma a/w total or near total loss of renal function

Clinical presentation: severe sepsis, with fever, chills, rigors, abdominal pain, shock, leucocytosis. Dipstick may be negative in complete obstruction

USS diagnosis of pyonephrosis dependent upon identification of internal echoes in dependent portion of kidney. Focal echopoor areas within parenchyma suggest destruction highly suggestive of pyonephrosis vs. infected hydronephrosis.

Management:

Antimicrobials and judicious drainage

Percutaneous vs. endoscopic drainage

Perinephric abscess

Pus within Gerota's fascia [cf. paranephric abscess = outside Gerota's fascia]

Routes of infection:

Kidney (60-80%)

ruptured cortical abscess (Staphylococcal seeding)

ruptured corticomedullary abscess (GNB)

ruptured calyx

pyonephrosis

calyceal diverticulum

staghorn

Haematogenous (10-30%)

Skin, mouth, lung infections

Infected perinephric haematoma

Paranephric space (10%)

Bowel (Crohns etc.)

Pancreas

Subphrenic/subhepatic abscess

Spine (Pott's etc.)

Clinical presentation

Insidious development of symptoms – 60% present > 14 days

Unexplained fever (absent in ~30%)

Nightsweats

Weight loss

Antalgic gait, flexion and external rotation with psoas irritation

Leucocytosis and pyuria in >75% cases

Multiple organisms = MSU and blood culture frequently miss organisms

Edelstein 1988 (therefore broad spectrum irrespective of results)

Diagnosis

AXR Normal in 40%

Loss of renal outline and/or psoas shadow

Scoliosis in up to 50%

Rarely gas or air/fluid levels

USS Generally hypoechoic with debris (occasionally air)

Hyperechoic thick, irregular wall

CT Investigation of choice

Management

(i) Appropriate antibiotics [NB. a number of reviews have shown that virtually all patients with acute pyelonephritis are rendered afebrile with 4 days of appropriate IV antibiotics (Thorley 1974, Fowler 1994) – thus if patient continues to spike after 4 days needs CT to exclude parenchymal or perinephric abscess]

(ii) Drainage (percutaneous or surgical)

Xanthogranulomatous pyelonephritis

Destruction of renal parenchyma with granulomatous infiltrates containing lipid-laden macrophages

Rare

Women > men

Adults > children

Peak incidence 40-60 yrs

Aetiology

- Unknown
- Nephrolithiasis in ~80%
- Upper tract obstruction +/- stones in others (PUJO, VUR)
- Increased risk in diabetes and immunocompromised

Presentation

- Fever
- Flank pain
- Weight loss
- Palpable mass
- Occasionally fistula

Pathology

Macro

- Hugely enlarged kidney with normal contour typical – occasionally focal
- Lesion starts in PC system and invades into renal parenchyma

Micro

- Lipid-laden (foamy) macrophages (difficult to distinguish from CCRCC on microscopy, and especially difficult on frozen section)
- Visible bacteria

Diagnosis

- Blood investigations as for pyelonephritis
- Positive urine cultures in 50-75%
 - Proteus > E. Coli
- Positive renal tissue culture in >90%

Radiology

- Classic triad in >50%

Unilateral renal enlargement

Non-function

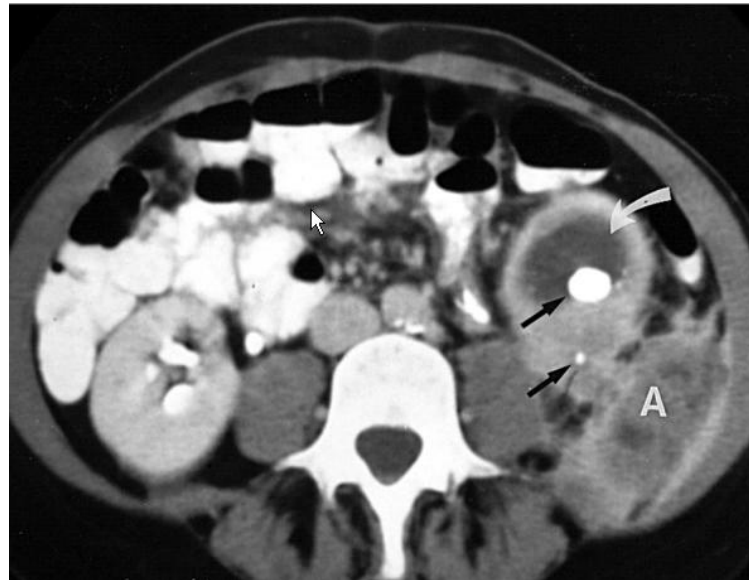
Calculus

- USS Hypoechoic centre and hyperechoic rim

- CT Investigation of choice

Enlarged kidney with normal contour

Water-density central lesion (often with stone at centre) surrounded by enhancing rim (differs from RCC)



Management

- Nephrectomy Rx of choice

- Establishes diagnosis

- Kidney often non-functioning

- Removes infective focus

- Antibiotics should be commenced ASAP and continued peri-operatively

- Partial nephrectomy may be an option for localised disease

- Conservative Mx reported but excluding malignancy difficult on Bx alone

Malakoplakia

= 'Soft plaque'

Originally described by Michaelis and Guttman in 1902

Chronic inflammatory condition affecting urinary tract believed to represent abnormal response to infection

Characterised by presence of lipid laden (foamy) macrophages containing pathognomonic Michaelis-Guttman bodies

Urinary tract involved in ~60% of cases (GIT, lung, skin and LNs also may be affected)

Rare

Female > males 4:1

Age > 50 yrs

Aetiology

- Unknown

- Coliform UTI (usually E. Coli) a consistent finding on MSU

- ~ 50% immunosuppressed or serious sytemic disease

- Believed to represent a failure of macrophages to adequately phagocytose bacteria

- Macrophages have reduced cGMP and increased alpha-1 antitrypsin

Presentation

- Usually patient has a history of recurrent coliform UTIs

- Bladder irritative LUTS and haematuria

- Ureter obstruction and haematuria

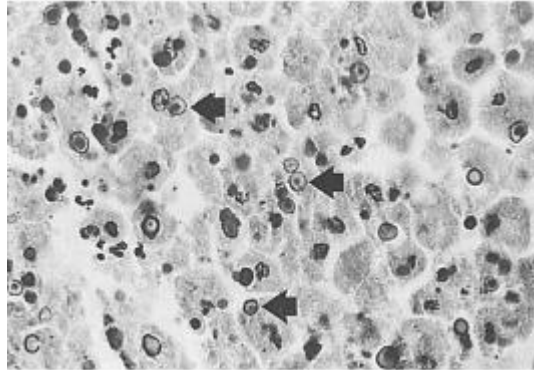
Kidney flank pain fever and mass
Abnormal mass on imaging
Rarely a/w renal vein thrombosis

Pathology

Soft yellow-brown plaques

Grow to form polypoid lesions

Micro Large histiocytes (vonHansellmann cells) containing basophilic inclusions (Michaelis-Guttman bodies – arrowed.



Diagnosis

Biopsy confirms diagnosis

Occasionally early disease a/w absence of MG bodies

IHC of histiocytes highly positive for alpha-1 AT which suggests diagnosis

Management

Chronic antibacterial prophylaxis

Rifampicin, doxycycline and TMP especially good as have intracellular activity

Nephrectomy for symptomatic unilateral lesions

Overall mortality 15% (~20% for renal disease)

Chronic pyelonephritis

Common

25% of ESRF population

Aetiology controversial. Some believe that untreated chronic infection – vasoconstriction (TXA2) – ischaemia – atrophy, but most believe that chronic infection alone is not sufficient to damage renal parenchyma.

Damage may have occurred in childhood with reflux of infected urine into kidney

Histology shows non-specific infiltrate of lymphocytes, plasma cells and occasionally PMNs.

Management – renal support and treatment of UTIs

Renal echinococcus

Extremely rare

Larva from dog tapeworm – human gut – duodenum penetration – liver – lungs. 3% pass through lungs to systemic circulation to kidneys

Typical renal hydatid cysts

Single in 95% of cases; bilateral in 5%

Slow growth – 1cm/yr

Triple epithelium – outer fibroblasts, middle capsule layer, inner germinal layer (producing more larva known as scoleces in daughter cysts) Daughter cysts detach from germinal layer to float freely within main cyst

Vague back pain, mass and haematuria

Predilection for seeding in renal poles

Diagnosis

<50% eosinophilia

Immunology variously reliable

Daughter cysts in urine diagnostic but usually not present

Management

Surgical

Avoid rupture – risk of anaphylaxis

Pretreat with mebendazole/albendazole

? inject scolicedal agent prior to removal

Mebendazole/albendazole unreliable as medical Rx alone